

Aortic Diameter and Pressure-Flow Sequence Identify Mechanism of Blood Flow During External Chest Compression in Dogs

ALAN D. GUERCI, MD, HENRY R. HALPERIN, MD, RAFAEL BEYAR, MD,
CHARLES BEATTIE, MD, PhD, JOSHUA E. TSITLIK, PhD, EDWARD C. WURMB, BS,
NISHA C. CHANDRA, MD, MYRON L. WEISFELDT, MD, FACC

Baltimore, Maryland

Aortic flow and pressure relations and aortic diameter were examined during sinus rhythm, internal cardiac massage, vest cardiopulmonary resuscitation, conventional manual cardiopulmonary resuscitation and high impulse manual cardiopulmonary resuscitation in 14 anesthetized large dogs. During sinus rhythm and during internal cardiac massage, ascending aortic flow and pressure increased simultaneously and the rise in ascending aorta pressure preceded the rise in descending aortic pressure by (mean \pm SEM) 28 ± 4 and 30 ± 1 ms, respectively. In contrast, during vest, conventional and high impulse cardiopulmonary resuscitation, ascending aortic flow lagged behind the initial rise in aortic pressure by 40 ± 4 to 46 ± 4 ms and ascending and descending aortic pressure increased simultaneously ($p < 0.001$ for each external compression mode versus sinus rhythm and internal massage). The ratio of

pulse pressure to stroke volume increased by an order of magnitude during all modes of external chest compression ($p < 0.001$ versus sinus rhythm and internal massage) and aortic diameter decreased during vest and high impulse cardiopulmonary resuscitation ($p < 0.05$ versus sinus rhythm and internal massage).

The hemodynamics of external chest compression depart from the normal physiologic sequence of stroke volume-induced increase in aortic pressure and diameter. The rise in aortic pressure precedes flow into the aorta, stroke volume does not fully account for pulse pressure, and aortic diameter decreases during chest compression. These data support the hypothesis that blood flow is due to fluctuations in intrathoracic pressure for high impulse as well as vest and conventional cardiopulmonary resuscitation.

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Despite application of increasingly sophisticated methods of analysis, the mechanism of blood flow during external chest compression remains controversial. Several lines of evidence support the hypothesis that blood flow results from a generalized increase in intrathoracic pressure. In animal studies (1-6), pressure changes of similar magnitude have been noted in several sites inside the thorax: right atrium, pulmonary artery, left ventricle, aorta, esophagus and lateral pleural space. Angiographic studies (2) have demonstrated that a column of blood extending from the left atrium to the aorta moves as a mass toward the periphery during chest

compression. Finally, with appropriate control of compression force and duration, studies (6) of compression rate and duration have established that blood flow and aortic diastolic pressure during manual chest compression are sensitive to compression duration but insensitive to compression rate. This result is opposite to that obtained with internal cardiac massage, in which aortic pressure and vital organ flow are sensitive to compression rate but not to compression duration.

Maier (7), Feneley (8) and their colleagues (7,8) have reported evidence of direct cardiac compression, particularly during high impulse manual chest compression. These data include a decrease of left ventricular dimension in the axis of compression, differences between intracardiac and pleural pressures and contrast echocardiographic evidence of transaortic flow during chest compression and transmitral flow during release of chest compression.

It is not clear whether small differences in intracardiac and pleural pressures during cardiopulmonary resuscitation are due to direct cardiac compression superimposed on a

From the Peter Belfer Laboratory for Myocardial Research, Divisions of Cardiology and Departments of Medicine and Anesthesiology, Johns Hopkins Hospital and Francis Scott Key Hospital, Baltimore, Maryland. This study was supported by Ischemic Heart Disease Specialized Center of Research Grant 5P50-HL-17655 and Grant R01 38174 from the National Heart, Lung, and Blood Institute, National Institutes of Health, Bethesda, Maryland.

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Address for reprints: Alan D. GuerCI, MD, Division of Cardiology, Carnegie 568, Johns Hopkins Hospital, Baltimore, Maryland 21205.

background of increased pleural pressure or inhomogeneities in intrathoracic pressure. Similarly, without measurements of changes in left ventricular volume, cardiac deformation does not by itself indicate the mechanism of blood flow. Because both mechanisms may be operative at the same time and because each mechanism has distinctive implications for optimization of vital organ perfusion (6), identification of the dominant mechanism of blood flow was attempted in these studies by measurement of aortic diameter and the sequence of aortic pressure and flow during conventional and high impulse manual external chest compression. In this analysis it was assumed that, as in a normally beating heart, under conditions of direct cardiac compression, aortic pressure would rise as a consequence of aortic flow, ascending aorta pressure would begin to rise before descending aortic pressure and aortic diameter would increase during chest compression. Alternatively, in a system of blood flow due to increased intrathoracic pressure, aortic flow would begin only after aortic pressure had risen to a level higher than extrathoracic arterial pressures, ascending and descending aorta pressure would increase simultaneously and aortic diameter would remain constant or even decrease as intrathoracic blood is extruded into the periphery. Compression with a pneumatic vest served as a prototype of blood flow due to changes in intrathoracic pressure, for the vest depresses the sternum by <1 cm, and when dogs of the size used in this study are supine, the anterior surface of the heart is 1 to 2 cm beneath the posterior surface of the sternum. Direct cardiac compression with the vest system used in this way is therefore highly unlikely.

Methods

Experimental preparation. Fourteen large mongrel dogs (22 to 28 kg) were anesthetized with an intravenous injection of 15 to 20 mg/kg body weight of the short-acting barbiturate, thiamyl sodium, followed immediately by intramuscular injection of 13.6 mg/kg chloralose and 136 mg/kg urethane. Animals were intubated endotracheally, ventilated with room air with use of a Harvard volume-cycled ventilator and given 500 to 1,000 ml of normal saline solution intravenously.

Two sets of experiments were performed, the first aimed at characterization of pressure and flow relations, the second at measurement of aortic diameter. In the pressure and flow studies ($n = 7$), a thoracotomy was performed in the fourth left intercostal space and a Biotronix BL5160 F11 circumferential flow probe was placed around the ascending aorta. Millar PC-470 micromanometers, with zero reference and calibration at 37°C, were advanced into mid-ascending and mid-descending aorta and right atrium through femoral cutdown and into the left ventricle from a brachial artery. Catheter position was confirmed by characteristic pressure tracings and by palpation before closure of the thoracotomy.

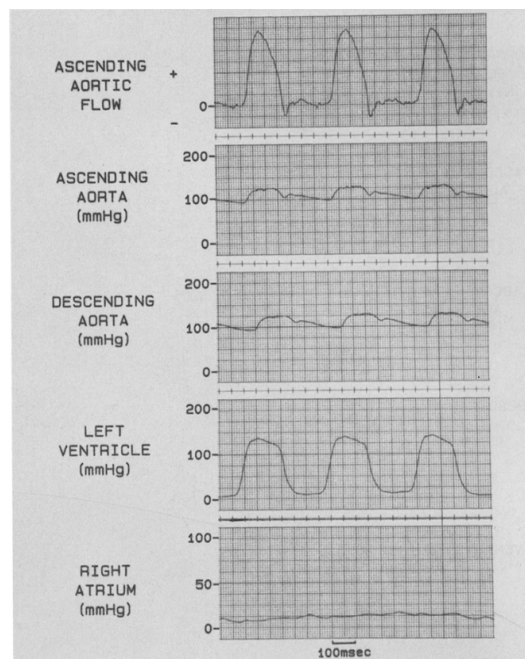


Figure 1. Pressure and flow recordings during sinus rhythm. Ascending aortic pressure rises synchronously with the onset of flow. Descending aortic pressure rises approximately 30 ms later.

After measurement of control pressures and flow (Fig. 1), the heart was fibrillated while the chest was still open by application of 60 Hz alternating current to a temporary pacing wire positioned in the right ventricle. Internal massage was performed for 30 to 60 s and the heart was then defibrillated. The chest was closed in layers and evacuated of air, and pressure and flow recordings were repeated during sinus rhythm.

External chest compression resuscitation. Ventricular fibrillation was induced for a second time and conventional external chest compression, high impulse external chest compression and vest cardiopulmonary resuscitation (Fig. 2) were performed in random order. Compression rates were 60/min for vest and conventional cardiopulmonary resuscitation, and both 60 and 120/min for high impulse cardiopulmonary resuscitation. High impulse cardiopulmonary resuscitation was performed as described by its originators (7), with an emphasis on a rapid, jabbing compression stroke. Conventional chest compression was performed according to old American Heart Association guidelines, at a rate of 60/min, with chest compression maintained for 500 ms and without special emphasis on the rapidity of the compression stroke (Fig. 3). One milligram of epinephrine was subsequently injected into the left ventricle, and conventional, high impulse and vest cardiopulmonary resuscitation were repeated.

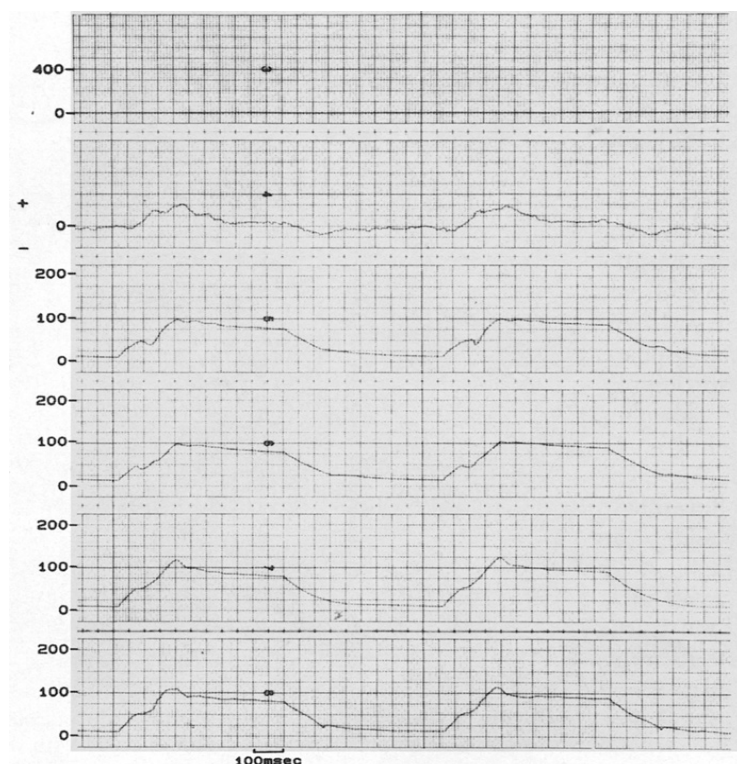


Figure 2. Pressure and flow recordings during vest cardiopulmonary resuscitation. All intrathoracic pressures rise simultaneously; flow begins approximately 45 ms later.

Conventional and high impulse cardiopulmonary resuscitations were performed with a hand-held precision force transducer (PCB Piezoelectronics 208A03) attached to a 5 × 8 cm hard rubber pad. The force transducer assembly was positioned directly over the heart and compressions were applied with a target force of 400 newtons (N). This level of force was selected because previous experience (6,9) has demonstrated that prolonged application of higher forces causes severe trauma to the sternum and rib cage. After the performance of external compression modes under the influence of epinephrine, the thoracotomy was opened rapidly and internal cardiac massage was repeated.

Each mode of cardiopulmonary resuscitation was performed until steady state pressures were reached, a process that usually required 20 to 30 s. Pressure and flow recordings were obtained at steady state, at a paper speed of 100 mm/s. Catheter position was confirmed by postmortem examination.

Radiographic studies. In the studies of aortic diameter ($n = 7$), pigtail catheters were positioned in the ascending aorta and right atrium through femoral cutdown. As in the pressure-flow studies, a transvenous pacing wire was also inserted into a femoral vein and advanced to the right ventricle. Posteroanterior and lateral cineangiograms were obtained at 60 frames/s during sinus rhythm with use of Renografin-76 and a General Electric MSI 850/Fluorocon 300 X-ray system. Ventricular fibrillation was then induced

electrically and cineangiograms were repeated for vest and high impulse cardiopulmonary resuscitation. High impulse cardiopulmonary resuscitation was performed by one of us (A.D.G.) standing next to the image intensifier. The radiographic studies also included one set of left ventriculograms performed in the same fashion in a dog in which the pigtail catheters were advanced into the left ventricle. The force transducer, which is radiopaque, was not used during the studies of aortic diameter. The chest was then opened and angiograms were obtained during internal cardiac massage.

Data analysis. In the pressure-flow studies, pressures and flow were recorded on a Gould Brush recorder. A Carolina FM 501 flow meter was used to measure the flow probe signal. Pressures and flows were also digitized in 2 ms intervals with use of a microcomputer-based data acquisition system (10). The moment of increase in pressure and flow was determined by consensus of visual assessment of high speed paper recordings and digitized records of the pressure and flow signals. Visual estimates of the timing of the onset of flow and the initial increase in pressure were reproducible to ≤ 5 ms.

In the studies of aortic diameter, aortic and right atrial pressures were recorded with Statham P23Db transducers and a Grass 7D recorder. In both the posteroanterior and lateral planes, diameters were measured as percent deviation from end-diastolic dimension.

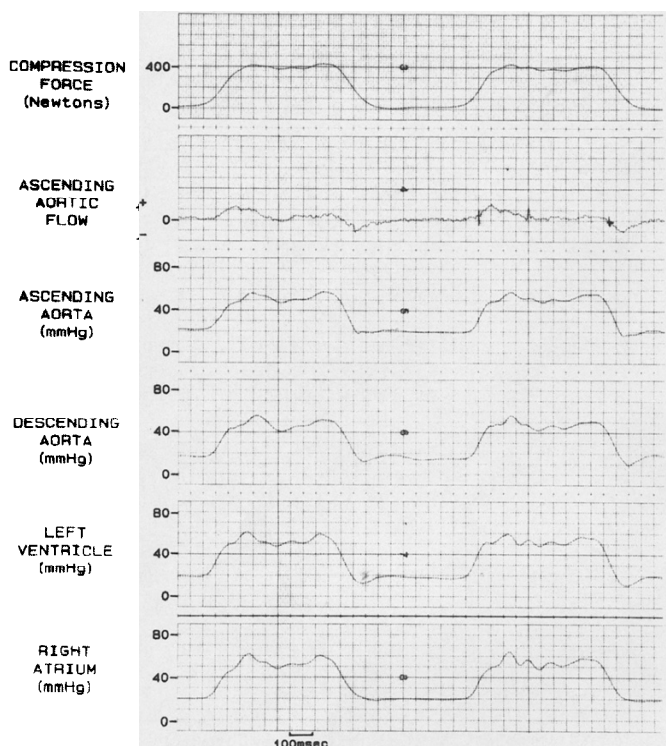


Figure 3. Pressure and flow during conventional external chest compression. Ascending and descending aortic pressures increase simultaneously. Flow begins 35 ms later.

Statistical analysis. Data were analyzed by *t* test with Bonferroni correction for multiple comparisons or by Newman-Keuls repeated measures analysis of variance. All data are presented as mean values \pm SEM.

Results

Aortic flow-pressure sequence (Table 1). The onset of aortic flow and the rise in aortic pressure occurred synchronously (-2 ± 2 to $+4 \pm 3$ ms, $p = \text{NS}$) during sinus rhythm and internal cardiac massage (Fig. 1). However, during vest, conventional and high impulse cardiopulmonary resuscitation, the onset of aortic flow lagged behind the initial rise in pressure by 40 ± 4 to 46 ± 4 ms ($p = \text{NS}$ versus each other, $p < 0.001$ for each mode versus sinus rhythm and internal massage (Fig. 2 to 4). This type of pressure-flow sequence, which violates the normal physiologic pattern of flow-induced increase in aortic pressure, was observed at equivalent compression force for conventional and high impulse cardiopulmonary resuscitation and before and after epinephrine.

Peak left ventricular pressure exceeded peak aortic pressure during vest cardiopulmonary resuscitation without epi-

nephine and high impulse cardiopulmonary resuscitation with and without epinephrine. The differences in peak left ventricular and aortic pressures during vest and high impulse cardiopulmonary resuscitation were attributable to a flail chest, which developed in one of the seven dogs studied and which may have allowed direct compression of the heart. In the remaining six dogs, including two others in which flail chest developed, there were no significant differences among peak left ventricular, ascending aortic, descending aortic and right atrial pressures during vest, conventional or high impulse cardiopulmonary resuscitation.

Whereas the rise in ascending aorta pressure preceded the rise in descending aortic pressure by 28 ± 4 and 30 ± 1 ms, respectively, during sinus rhythm and internal massage ($p = \text{NS}$), ascending and descending aorta pressures rose synchronously during vest, conventional and high impulse cardiopulmonary resuscitation ($p < 0.001$ for the latter three modalities versus sinus rhythm and internal massage).

Stroke volume and pulse pressure (Table 2). These were measured at steady state pressures for each modality. During sinus rhythm and internal massage, aortic pressure increased by 2.1 ± 0.2 mm Hg and 2.4 ± 0.1 mm Hg, respectively, for each milliliter of stroke volume ($p = \text{NS}$). In contrast, the ratio of pulse pressure to stroke volume was 5.4 to 12 times greater during vest (13.0 ± 0.8 mm Hg/ml), conventional (19.8 ± 4.2 mm Hg/ml) and high impulse cardiopulmonary resuscitation (28.8 ± 7.5 mm Hg/ml) ($p < 0.001$ for each versus sinus rhythm or internal massage). This finding, together with the observed decrease in aortic diameter (see later), suggests that ascending aorta pressure increased as a result of a compressive force acting directly on the thoracic aorta rather than as a result of filling of the aorta with blood from the left ventricle.

Aortic diameter (Table 3). Ascending and descending aorta diameter increased by $8 \pm 2\%$ and $13 \pm 3\%$, respectively, in posteroanterior and lateral projections during systole in sinus rhythm and internal massage. In contrast to this normal pattern of systolic aortic expansion, aortic diameter decreased by small but consistent amounts during chest compression in both the vest and high impulse modes. Ascending aorta diameter decreased by $7 \pm 2\%$ and $10 \pm 3\%$, respectively, during vest and high impulse cardiopulmonary resuscitation ($p < 0.05$ versus sinus rhythm or internal massage) and descending aorta diameter decreased by $4 \pm 3\%$ to $8 \pm 2\%$, respectively, ($p < 0.05$ versus sinus rhythm or internal massage) (Fig. 5). This reduction in aortic diameter occurred even in the presence of angiographic evidence of compression of the anteroapical region of the left ventricle by the sternum (Fig. 5).

Table 1. Intravascular Pressures and Aortic Pressure-Flow Relations During Sinus Rhythm and Cardiopulmonary Resuscitation (CPR) in Seven Dogs

Mode	Compression Force (N)	Left Ventricular Pressure (mm Hg)	Ascending Aortic Pressure (mm Hg)	Descending Aortic Pressure (mm Hg)	Right Atrial Pressure (mm Hg)	Q→P (ms)	Time Delay Asc Ao→Desc Ao (ms)
Sinus rhythm	—	119 ± 7/10 ± 1	121 ± 6/89 ± 5	121 ± 6/89 ± 5	7 ± 1	-2 ± 2‡	28 ± 2‡
Internal massage	—	81 ± 5/15 ± 2	74 ± 6/37 ± 5	71 ± 7/36 ± 5	26 ± 5/12 ± 2	4 ± 3‡	28 ± 4‡
Vest CPR	—	125 ± 7*/15 ± 1	116 ± 6*/26 ± 3	112 ± 6*/28 ± 3	117 ± 8/16 ± 4	-46 ± 4‡	0 ± 0‡
Conventional manual CPR	397 ± 7	41 ± 5/13 ± 1	42 ± 5/15 ± 2	42 ± 4/15 ± 2	41 ± 4/14 ± 1	-46 ± 4‡	2 ± 1‡
High impulse CPR (rate 60)	394 ± 6	71 ± 14*/12 ± 1	65 ± 8*/14 ± 2	56 ± 6*/15 ± 2	57 ± 14/10 ± 2	-41 ± 6‡	1 ± 1‡
High impulse CPR (rate 120)	391 ± 2	67 ± 4/11 ± 1	62 ± 3/14 ± 1	63 ± 3/14 ± 1	62 ± 2/12 ± 1	-42 ± 4‡	0 ± 0‡
Internal massage epinephrine	—	122 ± 4/15 ± 2	125 ± 3/73 ± 4	126 ± 4/72 ± 4	25 ± 5/13 ± 2	2 ± 2‡	30 ± 1*
Vest CPR, epinephrine	—	140 ± 16/18 ± 2	131 ± 13/38 ± 6	133 ± 14/38 ± 5	134 ± 5/14 ± 2	-46 ± 4‡	0 ± 0‡
Conventional manual CPR, epinephrine	398 ± 6	65 ± 16/11 ± 1	58 ± 10/21 ± 5	57 ± 11/22 ± 7	58 ± 11/11 ± 1	-43 ± 7‡	0 ± 0‡
High impulse CPR (rate 60), epinephrine	397 ± 9	84 ± 15*/11 ± 1	75 ± 9*/19 ± 4	70 ± 7*/21 ± 6	72 ± 12/14 ± 1	-40 ± 4‡	0 ± 0‡
High impulse CPR (rate 120), epinephrine	395 ± 1	71 ± 5/10 ± 1	67 ± 3/16 ± 2	66 ± 2/17 ± 2	65 ± 2/12 ± 1	-41 ± 4‡	0 ± 0‡

*Denotes peak left ventricular pressure greater than peak aortic pressure ($p < 0.05$). ‡p = NS versus other CPR modes with ‡ and $p < 0.001$ versus CPR modes with ‡. ‡p = NS versus other CPR modes with ‡. N = newtons; Q→P = interval between onset of ascending aortic flow and initial rise in aortic pressure. Minus sign indicates rise in pressure preceding onset of flow. Time delay refers to the interval between the upstroke of ascending aortic pressure and mid-thoracic descending aortic pressure. The onset of aortic flow occurred synchronously with the initial rise in aortic pressure during sinus rhythm and internal massage but lagged behind the initial rise in aortic pressure during vest CPR, conventional CPR and high impulse CPR ($p < 0.001$). The upstroke of descending aortic pressure preceded that of ascending aortic pressure (mean) by 28 ± 4 to 30 ± 1 ms during sinus rhythm and internal massage ($p = \text{NS}$), but these pressure upstrokes occurred synchronously during external modes of CPR ($p < 0.001$ versus sinus rhythm and internal massage). High impulse CPR was performed at a rate of 120 in only five of seven dogs. Comparisons between high impulse CPR at rate 120 and other modes of CPR are based on the same five animals.

Discussion

Hemodynamic patterns during cardiopulmonary resuscitation. Four hemodynamic patterns were observed during conventional and high impulse cardiopulmonary resuscitation in these studies: 1) aortic pressure increased before any detectable aortic flow, 2) ascending and descending aortic pressure increased simultaneously, 3) the ratio of pulse pressure to stroke volume increased by a factor of 5.4 to 12, and 4) aortic diameter decreased during chest compression. These patterns occurred at similar compression forces and, in all four instances, resembled vest cardiopulmonary resuscitation and differed significantly from pressure-flow relations and aortic dimensions during sinus rhythm or internal massage.

Two of these four hemodynamic patterns are, in fact, mechanistically indeterminate. Although ascending and descending aortic pressure increased simultaneously during high impulse as well as vest and conventional cardiopulmonary resuscitation, the initial upstroke in pressure may have been due to an increase in intrathoracic pressure that pre-

ceded direct cardiac compression. If direct compression did occur, delay in transmission of the stroke volume-induced pressure wave from the ascending to the descending aorta could have been obscured by maintenance of elevated intrathoracic pressure throughout the period of chest compression.

Pulse pressure-stroke volume relations: role of intrathoracic pressure. It is possible but more difficult to reconcile the relations between pulse pressure and stroke volume with direct cardiac compression. It is not likely that the small decreases in aortic diameter during external chest compression accounted for the roughly 10-fold increase in the ratio of pulse pressure to stroke volume, particularly because mean end-diastolic pressures were only 11 to 22 mm Hg during manual external chest compression, indicating that chest compression began when the aorta was on a compliant portion of its pressure-volume relation. Indeed, assuming that aortic capacitance remained constant during conventional and high impulse resuscitation, the stroke volume of 1.7 ml for each would have generated only a 4 mm Hg

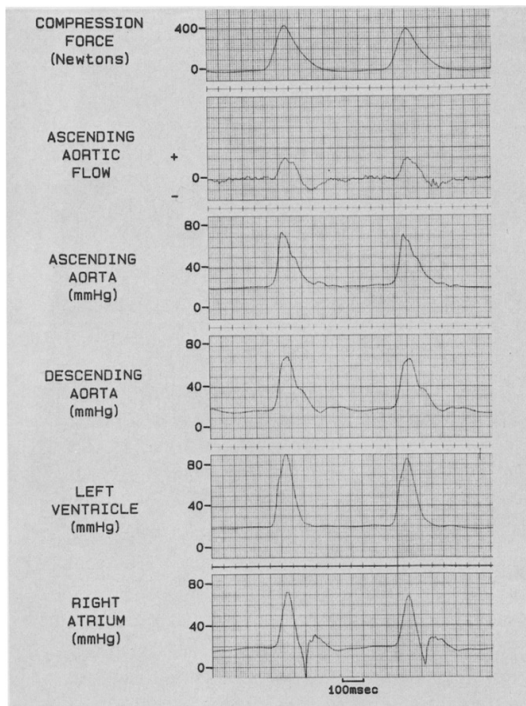


Figure 4. Pressure and flow recordings during high impulse cardiopulmonary resuscitation. Ascending and descending aorta pressures increase simultaneously; flow lags behind by approximately 40 ms.

increment in aortic pressure. This may have been the case, but this conclusion, that direct cardiac compression accounted for the stroke volume in these studies, leaves 80% to 90% of pulse pressure unexplained. Increased intrathoracic pressure may have been responsible for the balance of aortic pulse pressure, but this introduces an additional problem in the interpretation of these data. Changes in pleural pressure similar to those obtained in these studies are easily achieved during cough (11,12) and pressurization of isolated heart-lung preparations (13), in which direct cardiac compression is not possible. These same fluctuations in pleural pressure reproducibly generate the small stroke volume observed during manual chest compression in this study.

Although the explanation for the pressure-flow relations is arguable, the explanation for reduction in aortic diameter during vest and high impulse cardiopulmonary resuscitation is not. This decrease in aortic diameter has been observed previously (2) and is the result of a reduction in aortic volume, which in turn is due to extrusion of blood from the thoracic aorta to extrathoracic arteries by elevated intrathoracic pressure. Furthermore, the reduction in aortic diameter indicates that direct cardiac compression, if present, was subordinate to intrathoracic pressure as the mechanism of blood flow during high impulse cardiopulmonary resuscita-

Table 2. Stroke Volume and Pulse Pressure During Cardiopulmonary Resuscitation in Seven Dogs

	Pulse Pressure (mm Hg)	Stroke Volume (ml)	Ratio
Sinus rhythm	32 ± 2	16.1 ± 1.7	2.1 ± 0.2
Internal massage	36 ± 4	14.8 ± 1.5	2.4 ± 0.1
Vest CPR	90 ± 7	7.2 ± 2.1	13.0 ± 0.8
Conventional CPR	25 ± 4	1.7 ± 0.6	19.8 ± 4.2
High impulse CPR, rate 60	49 ± 9	1.7 ± 0.5	28.8 ± 7.5
High impulse CPR, rate 120	45 ± 8	1.8 ± 0.8	34.5 ± 9.0

Abbreviation as in Table 1.

tion. Therefore, the resemblance of aortic hemodynamics during high impulse and conventional cardiopulmonary resuscitation to those of vest cardiopulmonary resuscitation is not likely coincidental; intrathoracic pressure was the dominant mechanism of blood flow during high impulse and conventional cardiopulmonary resuscitation in these studies.

Role of peripheral vascular tone. Pulse pressure to stroke volume ratios are of necessity dependent on peripheral vascular tone. Therefore, choice of anesthetic agent may be critical to studies of this variable. Rapid completion of the study protocol is also important, because peripheral vascular resistance declines steadily during cardiac arrest (14). Neither of these potential problems affected the results of this study. An ultrashort-acting barbiturate (thiamyl sodium) was used to facilitate administration of the highly physiologic combination of chloralose and urethane (15). It is unlikely that substantial amounts of the former were still circulating by the time the experimental preparation was complete, because the values obtained for heart rate, stroke volume and blood pressure were within the normal range for conscious dogs (15). In all of the dogs studied, vest, conventional and high impulse cardiopulmonary resuscitation were applied within 3 to 4 min of arrest. Epinephrine was then administered, and these same modes of chest compression were again studied, all within the next 3 to 4 min. Therefore, the study protocol was completed quickly and the pulse pressure-stroke volume ratios were higher at the end of the

Table 3. Percent Change in Aortic Diameter During Cardiopulmonary Resuscitation in Seven Dogs

Mode	Asc Ao. PA	Asc Ao. Lat	Desc Ao. PA	Desc Ao. Lat
Sinus rhythm	+10 ± 2	+9 ± 3	+9 ± 2	+9 ± 2
Vest CPR	-7 ± 2	-7 ± 2	-8 ± 2	-6 ± 2
High impulse CPR	-8 ± 3	-10 ± 3	-4 ± 3	-5 ± 5
Internal massage	+8 ± 2	+8 ± 1	+11 ± 2	+13 ± 3

Asc Ao = ascending aorta; Desc Ao = descending aorta; Lat = lateral; PA = posteroanterior; other abbreviations as in Table 1.

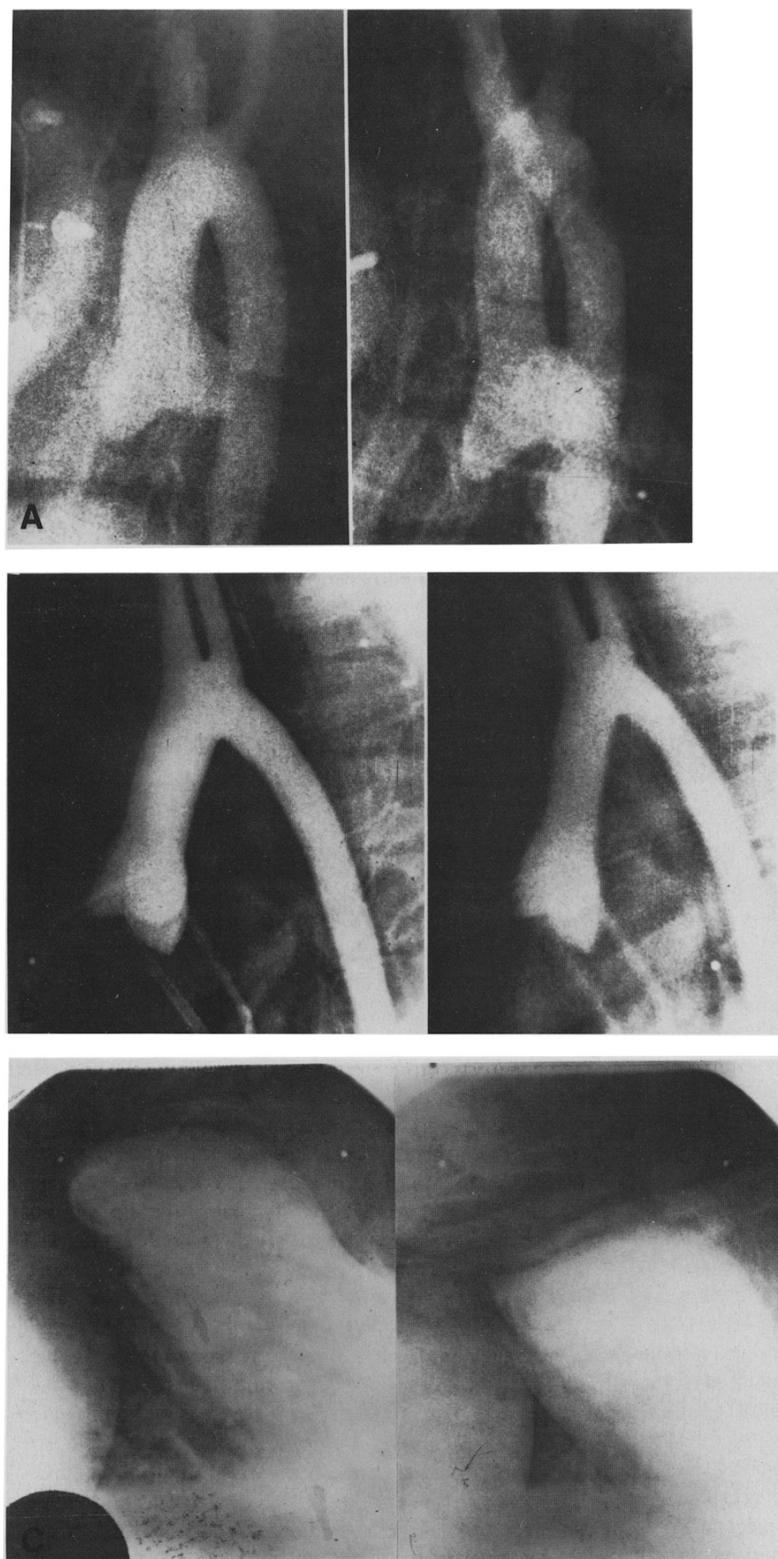


Figure 5. Aortic and left ventricular angiograms during high impulse cardiopulmonary resuscitation. The end of the release phase is at the left of each panel, the compression phase appears on the right. Ascending and descending aorta diameters decrease in posteroanterior (A) and lateral (B) projections despite indentation of the anterior surface of the left ventricle (C). In B, the sternum is on the left; in C, the sternum is at the top of the figure.

studies (after epinephrine) than at the beginning. This result indicates supernormal peripheral vascular resistance or decreased aortic capacitance, or both. Finally, insofar as unrecognized reduction of peripheral vascular resistance might have introduced artifacts into these pressure-flow relations, it should be understood that decreased peripheral resistance or increased aortic capacitance, or both, would be expected to reduce the pulse pressure-stroke volume ratio and slow the generation and propagation of a pressure wave. The unique features of external chest compression, as revealed by the analyses of this study, are the marked elevation of the pulse pressure-stroke volume ratio, the early rise in ascending aorta pressure and the simultaneous increase in ascending and descending aorta pressures—all directionally opposite from what would be expected in a pathologically relaxed vasculature.

Aortic flow, ascending aorta pressure and left ventricular pressure relations. Flow did not begin until left ventricular pressure was equal to or exceeded aortic pressure. This is a consequence of a basic physical principle—i.e., fluids will not move in the absence of a pressure gradient—and it is not evidence of direct cardiac compression superimposed on a background of small increases in intrathoracic pressure. Previous studies (1,6) have revealed that carotid artery flow begins early in the course of external chest compression. Therefore, ascending aorta flow begins as soon as thoracic aorta pressure exceeds extrathoracic arterial pressure and the inertial component resisting movement of blood is overcome. Because the thoracic aorta is vented to the periphery, aortic pressure may increase by slightly less than the increase in intrathoracic pressure. The aortic valve will open and ascending aorta flow will be appreciable only when left ventricular pressure exceeds aortic pressure.

The increase in ascending aorta pressure before the onset of aortic flow during external chest compression may be viewed as modest, generally 10 to 15 mm Hg. We suggest that this should be considered neither an artifact of delay in the flow meter circuitry (because pressure and flow increased simultaneously during sinus rhythm and internal massage) nor a weakness of the use of this variable as means of identifying the mechanism of blood flow. Even a modest rise in pressure, in the absence of flow, must be regarded as the result of a compressive force (pressure) acting on the aorta. Careful evaluation of the raw data (e.g., Fig. 4) reveals an even more startling result: aortic pressure reaches its peak level when only 1 or 2 ml has traversed the ascending aorta. A similar hemodynamic pattern can be seen in the original report of high impulse cardiopulmonary resuscitation (7). Although the separate montage of pressure and flow graphics in the report by Maier et al. (7) renders analysis of the timing of pressure and flow difficult (e.g., their Fig. 4), it is clear that very high aortic pressures are developed in association with very small stroke volumes. In their Figure 6 (7), for example, aortic pressure exceeds 100 mm Hg when

only a small fraction of stroke volume has been ejected. Mean stroke volume for high impulse cardiopulmonary resuscitation was only approximately 6 ml in their study (7) (their Table 2).

It may be argued that higher compression forces than those used in our studies would have further displaced the sternum, producing direct cardiac compression. However, in the aortic pressure-flow studies, three of seven dogs developed a flail chest by the end of the experiment, and one other study, not included in this report, was abandoned because flail chest occurred within 30 s of the initiation of manual chest compression. Higher compression forces would undoubtedly have caused even more severe chest trauma. Hemodynamic information obtained under these conditions would be of questionable relevance to cardiopulmonary resuscitation in humans or dogs.

Comparison of high impulse, conventional and vest cardiopulmonary resuscitation. Intrathoracic pressures were significantly higher during high impulse cardiopulmonary resuscitation than during conventional cardiopulmonary resuscitation. This may be due to more effective trapping of air in the lungs with the rapid compression stroke of the high impulse technique (16). Of note, however, stroke volume was similar for both high impulse and manual cardiopulmonary resuscitation despite the difference in peak aortic pressure. This equivalence of stroke volume was the result of the prolonged period of elevated intrathoracic pressure and blood flow during conventional cardiopulmonary resuscitation (6).

One common characteristic of cough, vest cardiopulmonary resuscitation, and manual external chest compression in humans as well as in large dogs is a wide pulse pressure and a low aortic diastolic pressure (6-8,10,11,17-19). For the reasons already discussed, this feature of aortic pressure does not prove that blood flow during conventional manual chest compression or high impulse compression is due to elevation of intrathoracic pressure; a small amount of direct cardiac compression may occur against a background of substantial elevation of intrathoracic pressure. At the same time, these characteristics of aortic pressure, together with the relation between stroke volume and aortic pulse pressure in this study, suggest that stroke volume does not play a primary role in the generation of aortic pulse pressure during chest compression. Furthermore, because elevation of intrathoracic pressure can cause blood flow (6,11-13), interpretation of small differences in intrathoracic pressure becomes problematic.

It may be that some factor related to animal size accounts for discrepant results of high impulse cardiopulmonary resuscitation in different laboratories. The repeated generation of left heart pressures in excess of 100 mm Hg by the developers of this technique, as opposed to excessive chest trauma at lower pressures in our hands, suggests that this is so.

Implications. Although limited in quantity and scope, hemodynamic data from humans during external chest compression resemble those of cough: equivalence of right and left sided pressure, wide pulse pressure and low diastolic arterial pressure. Wide pulse pressure and low diastolic pressure are characteristic of the original work of Maier et al. (7, their Figure 11) as well as other studies (17-20). Together with the dependence of arterial pressure and flow in humans on compression duration rather than compression rate (6,21) and echocardiographic evidence of mitral valve opening during chest compression (22,23), available data indicate that fluctuations in intrathoracic pressure account for blood flow during conventional external chest compression in humans. Although the present canine studies are only of heuristic value in relation to the physiology of high impulse chest compression in humans, they suggest that the high impulse technique does not differ significantly from other forms of external chest compression.

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